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# Implications of biological findings for psychological treatments of post-traumatic stress disorder

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Psychological research conducted on post-traumatic stress disorder (PTSD) over the past two decades has focused principally on two goals: (i) estimating prevalence of PTSD following a variety of traumatic exposures and (ii) identifying diagnostic instruments and effective treatments. Studies designed to understand the biological and psychological underpinnings of the disorder (psychopathology research) paralleled these efforts. Although biological research on PTSD is still in its nascent stages of development, it has progressed significantly to begin to offer important insights into the etiology, diagnosis, and treatment of the disorder.

Despite the possibility for collaboration, few efforts at integrating biological findings and psychological treatment development are apparent in the literature. The focus of this paper is to begin a dialogue among those conducting research on the biological aspects of PTSD and those professionals concerned with developing effective psychological treatments for PTSD. We begin with a basic overview of important biological findings in PTSD, discuss the potential clinical implications of these findings, and then move to a discussion of psychological treatments that have been found to be effective for PTSD. Our focus is to integrate extant findings on treatment efficacy with biological parameters known to be dysregulated in PTSD patients. Finally, we explore future directions for treatment development for PTSD, suggesting how innovative treatments may successfully address aspects of the biological dysregulation that accompanies PTSD.

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## Biological findings associated with PTSD

Biological research on PTSD is a growing field offering much promise. With high prevalence rates of PTSD internationally, the development of an array of treatment approaches that can be used by clinicians remains a high priority for those concerned with national and international public health. Psychological treatments are presently acknowledged as first-line treatments of choice for PTSD [1], yet the outcomes of treatment need improvement. There is no single treatment that can be universally applied that achieves consistently positive outcomes. More work is needed. As such, clinicians and researchers interested in the psychological treatments for PTSD will likely benefit from an understanding of core biological findings associated with PTSD and their possible clinical implications. Because it is beyond the scope of this article to comprehensively review all findings in this literature, we focus on those that are the most consistent and that appear to have implications for psychological treatment development.

### *Psychophysiological findings*

#### *Primary findings*

Research on psychophysiologic arousal associated with PTSD dates to Kardiner's defining of "traumatic neuroses" in 1941 [2]. Early findings of arousal of combat veterans to trauma-related cues influenced our definition of PTSD, and hypervigilance, physiological reactivity, and enhanced startle are viewed as hallmark features of the disorder. One of the most consistent findings is that individuals with PTSD show heightened physiologic arousal when exposed to trauma-related cues. For example, Keane et al. (1998) found that PTSD combat veterans demonstrated elevated psychophysiologic arousal and reactivity when presented with audiovisual and imagery-based cues of combat experiences [3]. These findings have also been replicated with other traumatized populations, such as child sexual abuse [4] and motor vehicle accident survivors [5].

Prins et al. note that psychophysiologic reactivity is not consistently observed across all those with PTSD [6]. Griffin et al., in a study of survivors of rape, found that dissociation may be at least partly responsible for this individual difference [7]. Their research indicated that participants with high levels of dissociation, when asked to verbally discuss their sexual assault, showed a decrease in skin conductance and heart rate rather than an increase in physiologic reactivity for those with lower levels of dissociation. In addition, this lack of physiologic response by all PTSD subjects could be a function of the inaccuracy of diagnostic procedures or the existence of specific subtypes of the disorder for which there is an absence of physiologic reactivity. Yet, it is likely that a subgroup of PTSD patients may dissociate when exposed to trauma cues and thus require specific interventions to address this psychological response.

There are some conflicting findings regarding whether baseline heart rate elevations are associated with PTSD. When compared with control subjects, subjects with PTSD have consistently higher heart rates [8]. However, when compared with subjects with other anxiety disorders or trauma-exposed populations without PTSD, the results have not been as definitive [9]. A recent meta-analysis by Buckley and Kaloupek indicates that PTSD is associated with elevated levels of basal heart rate and diastolic blood pressure, even when compared with a traumatized, non-PTSD comparison group [10]. Additionally, they concluded that differences in resting heart rate could not be attributed solely to anticipatory anxiety. This meta-analysis also explored whether the chronicity of PTSD is associated with poor cardiovascular health. Buckley and Kaloupek [10] compared studies with more chronic PTSD participants (12 or more years post-trauma) with those with more recently traumatized participants (less than 8 years). More chronic PTSD samples had larger effect sizes for heart rate differences than samples of more recent PTSD cases. Although preliminary, these findings may suggest that individuals with PTSD demonstrate elevated basal cardiovascular activity, findings that might result from an adaptation over time to a chronic stress condition.

Finally, there is preliminary evidence that the initial psychophysiologic response to trauma exposure may be a predictor of PTSD development. Shalev et al. found that higher heart rate 1 week after a motor vehicle accident predicted PTSD severity 4 months after the event [11]. Yet, heart rate at 1 month and 4 months post-trauma exposure did not. Although future research exploring the generalizability of these results is necessary, these findings suggest that immediate physiologic response to trauma exposure may be a marker for the eventual development of PTSD.

*Clinical implications*

These physiologic findings have several possible implications for psychological treatments. First, hyperarousal to trauma-related cues supports the utility of an exposure therapy paradigm. Research across anxiety disorders indicates that those with greater initial reactivity to anxiety-eliciting cues respond best to exposure therapy approaches. However, as we have noted, not all individuals demonstrate this heightened arousal. This suggests that there may be further subtypes of the disorder. If so, the subtypes may respond differently to various treatment approaches. More specifically, one can argue that those without physiologic arousal to trauma-related cues are not good candidates for exposure therapy. This is clearly an empirical question. Rather, these nonreactors may be particularly well suited for cognitive or interpersonally oriented treatment approaches. Furthermore, if the lack of arousal is caused by dissociation, clinical emphasis on grounding and mindfulness training may be warranted before beginning a course of exposure therapy.

Second, the results from Buckley and Kaloupek's meta-analysis [10] suggest that treatments targeting basal heart rate elevations may be important.

These observations support the use of exposure therapy, relaxation training, and stress management techniques broadly (i.e., cued relaxation, breathing retraining, etc.). Another important consideration concerns the related health risks associated with this sustained hyperarousal. Comprehensive treatment programs for PTSD might include wellness groups focused on healthy living and other behavioral medicine interventions that would decrease the likelihood of physical illness.

Third, Shalev et al.'s findings [11] suggest that early physiologic arousal may be a risk factor for PTSD development. If replicated, clinical research should examine the effectiveness of relaxation training and behavioral intervention techniques targeting this at-risk population. Foa et al. [12] and Bryant et al. [13] offer models for early intervention using cognitive–behavioral strategies.

### *Neuroendocrine system*

#### *Primary findings*

Previous research has found that the neuroendocrine system plays an important role in the normal stress response [14]. Researchers interested in trauma and PTSD consequently began to examine this system, with a focus on the glucocorticoids and catecholamines that are normally released during stress. It was expected that traumatized individuals with PTSD would have higher levels of glucocorticoids (e.g., cortisol) because of their often chronic states of stress. However, findings by Yehuda et al. suggest just the opposite: PTSD is associated with lower baseline levels of cortisol [15] and greater number of glucocorticoid receptors [16] (refer to Pitman and Orr for contrary findings [17]). On the basis of this research and other related work, Yehuda has suggested that PTSD is not simply a chronic stress response but instead is indicative of a system that is perhaps maximally responsive to stress [18].

Two interesting longitudinal studies offer additional information on the relationship between cortisol levels and PTSD. First, motor vehicle accident victims who developed PTSD within 6 months of the accident had lower levels of cortisol immediately following the traumatic event than individuals who did not develop PTSD [19]. Second, when tested immediately following a rape, women with a history of a prior rape or assault had lower levels of cortisol than women with no such history [20]. This latter result is consistent with the animal literature that suggests that exposure to a stressor may change the response of the HPA axis to later stressors [21].

A second line of research has focused on the catecholamines released during stress. The catecholamines increase heart rate and blood pressure and are associated with enhanced memory, hypervigilance, fight-or-flight responses, and level of alertness [22]. Kosten et al. found higher levels of epinephrine and norepinephrine in combat veterans with PTSD when compared with veterans with schizophrenia or major depression [23], suggesting that

increased levels of catecholamines may be responsible for certain PTSD-specific symptoms.

Complementary work by Perry et al. [24] examined the number of alpha-2-adrenergic receptor sites, whereas Southwick et al. [25] measured behavioral responses to alpha-2-adrenergic antagonists. Both combat veterans and children diagnosed with PTSD had fewer alpha-2-adrenergic receptor binding sites per platelet than did control subjects [24], as would be expected on the basis of the higher levels of circulating catecholamines found by Kosten et al. [23]. This finding has also been replicated when PTSD patients were compared with those diagnosed with generalized anxiety disorder and depression [26]. Furthermore, the introduction of yohimbine, an antagonist that blocks the alpha-2-adrenergic receptor sites, resulted in panic attacks for 70% of the veterans with PTSD and flashbacks for 40% of these patients [25], indicating that increased levels of catecholamines may influence the expression of PTSD symptoms.

*Clinical implications*

The directional nature of the relationship between cortisol levels and PTSD has not been determined, but prospective research in this area will permit a more careful analysis of cause and effect. Recommendations for psychological treatment will depend on clarification of this association. If, for example, low cortisol levels are precursors to PTSD and serve as risk factors, early interventions focusing on the acquisition of anxiety management and relaxation skills may be appropriate.

Research supporting an elevation of catecholamines in PTSD patients suggests that exposure therapy, relaxation training, and stress management groups may be especially beneficial for addressing the hypervigilance and hyperarousal associated with high levels of epinephrine and norepinephrine. However, the challenge research (i.e., yohimbine) by Southwick et al. [25] suggests that there are clear individual differences within PTSD samples that may represent unidentified subtypes of the disorder. It is unclear at this point whether responses to challenge tasks may be one marker for distinguishing between these subtypes, but further research into this area is recommended. Furthermore, the relationship between PTSD, flashbacks, and panic attacks suggested by the challenge research may speak to our need to develop interventions that target comorbid conditions, such as PTSD and Panic Disorder.

*Hippocampal volume*

*Primary findings*

Infra-human research indicates that chronic stress is associated with hippocampal atrophy [27]. In exploring possible structural changes associated with PTSD in humans, initial studies by Bremner et al. found that those with combat-related PTSD had smaller right-side hippocampal volume when compared with veterans without PTSD [28]. Gurvits et al., however,

did not replicate these findings. Instead, they reported bilateral hippocampal reductions when combat veterans with PTSD were compared with combat veterans without PTSD [29]. Bremner et al. [30] and Stein et al. [31] found smaller left-sided hippocampal volume in samples of adults with a history of child sexual abuse. In contrast, De Bellis et al. [32] reported that sexually abused children did not show localized hippocampal damage but did show generalized brain atrophy. Overall, the above findings suggest that smaller hippocampal volume is associated with PTSD, but it is premature at this time to draw conclusions on the basis of the specific location of these volumetric differences. It is also not at all clear that the differences observed are consequences of trauma exposure or PTSD. They could be risk factors. Only prospective studies will inform us about causality.

*Clinical implications*

Although preliminary, the findings regarding hippocampal volume may have clinical utility, regardless of the directionality of the relationship. Because the hippocampus is involved with memory, smaller hippocampal volume may have implications for basic information processing, attention, treatment adherence, and homework compliance. Interventions that provide patients with structure and compensatory strategies (e.g., note-taking, writing down homework assignments) to address short-term memory defects may improve patients' engagement in the therapeutic process. Research on the functions of the hippocampus also suggests that therapy focusing on integrating verbal and visual memory and placing memory into context may be especially beneficial. Borrowing strategies from cognitive rehabilitation models may ultimately benefit the PTSD patient.

*Event-related electroencephalographic potentials*

*Primary findings*

To examine central nervous system responses in PTSD, researchers have studied event-related electroencephalographic potentials (ERPs). ERPs are averages of the EEG response to a particular stimulus, resulting in a waveform. ERPs are evaluated according to the direction (Positive [P] or Negative [N]) and temporal placement of the response (e.g., number of milliseconds after the stimuli are presented). For example, when speaking of a P2 response, researchers are referring to an ERP that is positive in valence and occurs approximately 200 milliseconds after a stimulus is presented. Although this research is in its early stages, there are several important findings to consider.

First, the research indicates that combat veterans with PTSD have a unique response when exposed to stimuli that increase in intensity. Whereas most individuals show increased P2 amplitudes to progressively louder stimuli, Paige et al. found that 75% of veterans diagnosed with PTSD actually show decreasing amplitudes to such stimuli; of the non-PTSD veterans, 83% showed the more typical pattern, with increasing P2 amplitudes to more

intense stimuli [33]. Although these findings need replication across populations, they do suggest a CNS response reflecting an effort to dampen aversive auditory stimuli.

A second area of research has revealed that combat veterans demonstrate less habituation to recurring stimuli. In this research paradigm, veterans are exposed to a pair of auditory clicks. Studies in control samples demonstrate reliably that the ERP response to the second stimulus is typically smaller. In contrast, PTSD veterans demonstrate less habituation to the second click when compared with combat-exposed, non-PTSD veterans [34,35]. The authors speculated that the lack of habituation may reflect underlying hyperarousal and be manifested in hypervigilant behaviors.

Finally, a third area of research has explored P3 responses using the auditory oddball paradigm, in which subjects are asked to pick out target stimuli (presented infrequently) among a series of distracters. The reduction in P3 amplitude to target stimuli is not specific to PTSD, so this area of research is not reviewed here. The most relevant finding at P3 for PTSD complements the psychophysiologic research described earlier. When the oddball paradigm was modified to include trauma-specific or novel stimuli as distracters, PTSD veterans allocated more attention to the distracting stimulus than did non-PTSD veterans [36].

*Clinical implications*

There may be important clinical implications that come from this research, but to date, these implications are merely speculative. The ERP data suggest that PTSD patients respond differently to certain stimuli than do controls, which indicates a particular sensitivity to trauma-specific or novel stimuli. The data also may indicate a tendency of PTSD patients to use avoidance when presented with aversive stimuli. These implications are consistent with other lines of research demonstrating hypervigilance, hyperarousal, and avoidance associated with the disorder. Future clinical research may benefit from the inclusion of ERPs in outcome studies to provide us with important information regarding the clinical relevance of the ERP findings. If preliminary interpretations of the ERP data are supported by more extensive research, treatments targeting the seemingly automatic response to avoid intense stimuli should be evaluated. Additionally, PTSD patients' tendency to selectively attend to novel, distracting, and/or trauma-specific information may suggest that PTSD patients will miss relevant information in their efforts to detect threat-related stimuli. Treatment aimed at aiding patients in identifying and effectively evaluating threat may be worth further investigation.

*Sleep research*

*Primary findings*

Primary symptoms of PTSD include sleep disturbance and nightmares. Research using polysomnography offers objective evidence of sleep-related

disturbances associated with PTSD but is limited by findings that are based mostly on combat veterans and small sample sizes. Despite these limitations and the existence of some contradictory findings [37], sleep studies offer much needed information about the nature of sleep disturbances associated with the development and maintenance of PTSD. There is preliminary evidence, for example, that PTSD subjects have less REM sleep than depressed subjects [38]. In addition, studies by Lavie et al. [39] and Mellman [40] suggest that PTSD is associated with increased awakenings and motor activity and decreased sleep time. Additionally, Dagan et al. found that PTSD patients had a higher threshold for arousal from non-REM sleep [41], suggesting a paradoxical deepening of sleep associated with PTSD. Finally, research on nightmares is limited but does suggest that repetitive nightmares that recapitulate traumatic events are specifically associated with PTSD [40,42].

*Clinical implications*

On the basis of the research described above, there are clinical implications for the psychological treatment of insomnia in PTSD. Comprehensive treatment of PTSD should include a component addressing insomnia. Likewise, focusing treatment on PTSD-related nightmares ought to be an important complement to other treatment strategies. A significant body of research indicates that nightmares can often be treated effectively, both in individuals with and without PTSD (reviewed below).

*Limitations of extant biological research*

Although the above-mentioned literature is clearly relevant to clinical research on PTSD, much is still unknown in terms of the biological mechanisms associated with PTSD. First, even the findings with the most empirical evidence are in need of further support via replication across laboratories and targeted populations, and with larger, diverse samples. Second, the literature to date contains instances of contradictory findings, and, therefore, conclusions made on the basis of this research must be considered tentative. Most findings derive from cross-sectional studies and do not permit an analysis of cause and effect. Moreover, the extent to which biological deviations are corrected by effective therapies has not yet been explored. Finally, although statistically significant differences have been reported, the actual clinical significance of these differences is unknown in most cases. Studies relating those findings at the cellular and systems levels of analysis to the behavioral/symptom level are few indeed. More work on the relationships across these levels is needed.

## Psychological treatment for PTSD

There are a number of potential ways in which biological findings can inform the treatment of PTSD. Some of these issues are likely addressed by current PTSD treatments, but others are not and can thus inform future

treatment development. We begin by reviewing those treatment approaches with the most empiric support, concluding with future treatment directions that may address some of the limitations of current approaches. Discussion of cognitive-behavioral and other innovative treatments of PTSD address the biological findings reviewed above.

## *Empirically validated treatment approaches*

### *Exposure therapy*

Exposure therapy has one of the longest traditions in the psychological treatment of PTSD. Exposure therapy for PTSD is implemented in two ways: in vivo exposure or imaginal exposure. In vivo exposure involves presenting the client with physical cues related to the trauma (e.g., location of the trauma, objects or conditions associated with the trauma) to reduce avoidance and promote habituation of emotional responding to these conditioned cues. Theoretically similar, imaginal exposure is used in the treatment of PTSD when in vivo exposure is not possible or feasible because of the trauma occurring much earlier in life or in an inaccessible location. Cues are presented in imagery in an effort to describe details of an event or set of events while promoting contact with affect associated with the traumatic event(s). As with in vivo exposure, the goals of imaginal exposure are the reduction of avoidance and habituation of emotional responding.

Exposure therapy was first used by Keane et al. to treat PTSD associated with traumatic combat events in veterans [43–45]. Significant reductions in trauma symptoms, anxiety, and other related symptoms were noted as a function of these interventions. Following this preliminary work, imagery-based exposure therapy was found to be superior to a wait-list condition on standard psychometrics and clinician ratings of symptoms, both at post-treatment and 6-month follow-up [46], and the addition of exposure therapy to available treatments of PTSD was found to improve overall outcome for patients [47,48]. Expanding this work to rape survivors, Foa et al. [49] compared exposure therapy with stress inoculation treatment (SIT; an anxiety management condition), supportive counseling, and a wait-list control, measuring outcomes by clinical ratings of symptoms and standardized psychometrics. Although SIT was superior to the counseling and wait-list conditions at post-treatment, those patients who received exposure therapy performed best on measures of PTSD at 3.5-month follow-up when compared with all other conditions. More recently, the utility of exposure therapy has been demonstrated in the treatment of clients with PTSD stemming from many different traumatic events [50–52], further extending the results found with combat veterans and rape survivors. In other randomized controlled clinical trials, exposure therapy was also equally effective as cognitive therapy for PTSD, the combination of cognitive therapy and exposure therapy, anxiety management training, and the combination of anxiety management and exposure therapy [50,52,53].

In summary, the extant data support the use of exposure therapy in the treatment of PTSD [54]. In addition, a review of the existing biological findings associated with PTSD supports exposure therapy's emphasis on the integration of visual and verbal memories, habituation to presented stimuli, and extinction of physiological responding to trauma-related triggers. Because exposure therapy may be likely to influence some of the biological correlates of PTSD, future research on exposure therapy should also evaluate biological markers of PTSD pre- and post-treatment to enhance our understanding of the interplay between biology and treatment and our overall understanding of the disorder itself. Although it is clear that exposure therapy is an effective treatment for PTSD, not all patients respond positively to this treatment [55]. Clinical researchers may benefit from the use of biological indictors (e.g., physiologic measures, cortisol levels, ERP responses) to identify commonalities between those who do and do not respond to this treatment approach to help facilitate the matching of patients to the most appropriate treatments.

*Anxiety management training*

Efficacy trials indicate that anxiety management training (AMT) leads to reductions in PTSD symptoms and improved psychosocial adjustment. AMT typically involves teaching patients an assortment of behavioral and cognitive strategies to enhance their capacity to manage the emotional responses associated with PTSD. Such skills might include relaxation training, breathing retraining, trauma education, cognitive restructuring, or communication skills training. In addition, some programs for PTSD emphasize the incorporation of anger management training [46,56], given the frequency with which anger is a problem in this population. In some studies [49,53], AMT has been compared with exposure therapy and found to result in significant reductions in symptoms for female rape survivors, although the long-term effects were not as strong as exposure therapy. However, in another study comparing exposure therapy with AMT, AMT was found to result in significant treatment attrition [46]. Treatments focusing on one component of AMT, such as biofeedback-assisted relaxation treatment [57] or AMT focused on anger and rage [56], have also received preliminary empirical support.

In summary, although the data on AMT are not as clear or as consistent as the data for exposure therapy, there is evidence to suggest that a skills training approach, such as AMT, can have a favorable impact on the symptoms of PTSD. In particular, the psychophysiologic arousal reviewed earlier may be aided especially by some components of AMT, such as relaxation skills training. Furthermore, cognitive restructuring that includes a focus on defining and evaluating risk may have an impact on PTSD patients' tendency to selectively attend to potentially threatening stimuli. Therapy process research, exploring the impact of AMT on the relevant biological markers, will allow us to test these face valid predictions empirically.

*Combined treatment approaches*

Given the findings described above for exposure therapy and symptom management treatments, it can be argued that the approaches to psychotherapy for PTSD with the most potential may be those that combine components of exposure therapy, cognitive therapy, and anxiety management training. Combination treatments that include an array of cognitive-behavioral strategies have the advantage of addressing multiple problems while incorporating clinical techniques that have considerable empirical support. One of these package treatments was described by Keane et al. [58] as employing a phase-oriented approach to treating severe and chronic PTSD including the following six phases: (i) behavioral stabilization, (ii) trauma education, (iii) anxiety management skills, (iv) trauma focus work, (v) relapse prevention skills, and (vi) aftercare procedures. In fact, testing such a phased approach with motor vehicle accident survivors, Fecteau and Nicki [59] found clinically significant results on clinical ratings, self-report questionnaires, and psychophysiologic measures at post-treatment and 6-month follow-up, compared with a wait-list control group. Frueh et al. [60] also assembled a multi-component treatment for combat-related PTSD that combined exposure therapy, AMT, and cognitive therapy, with preliminary positive treatment effects.

One of the most widely used combination treatments is the multidimensional treatment package developed by Resick and Schnicke [61] for rape-related PTSD, Cognitive Processing Therapy (CPT). Combining elements of exposure therapy, AMT, and cognitive restructuring, CPT was far superior to a wait-list comparison group at post-treatment, 3-month, and 6-month follow-up on clinician ratings and psychometric inventories of PTSD. A recent study comparing CPT and exposure therapy for rape-related PTSD found that the two treatments seemed to be equally effective and more effective than a wait-list control condition, although the CPT treatment also seemed to reduce comorbid symptoms of depression more effectively [62].

Another, more controversial, treatment of PTSD is Eye Movement Desensitization and Reprocessing (EMDR) [63,64], which combines components of exposure therapy and cognitive therapy with repeated sets of lateral eye movements. Although EMDR has garnered some empirical support for the treatment of PTSD, there is no study supporting EMDR as a treatment superior to any of the existing treatments for PTSD, such as exposure therapy, anxiety management training, or cognitive therapy [54]. In addition, its mechanism of action is not based on any contemporary theories of human behavior, learning, or cognitive science, although some discussion of Pavlovian neurophysiology is provided across publications [63,64]. For these reasons, EMDR is probably best conceptualized as a combined treatment approach for PTSD with some empirical support but in need of further research to determine its active ingredients and to distinguish its efficacy as separate from the exposure-based or cognitive therapy components it contains.

Overall, a combined approach to treatment is consistent with the findings in the biological literature that indicate heightened physiologic arousal, nightmares, and selective attention to trauma-related stimuli. Research suggesting smaller hippocampal volume associated with PTSD also supports the use of trauma-focused components emphasizing integration of verbal and visual memory and contextualization of traumatic memories. In addition to the existing components of treatment, clinicians may want to consider how specific deficits, such as short-term attention difficulties, may affect the application of this treatment approach and adjust treatment strategies accordingly. Behavior that may be interpreted as noncompliance or resistance could also be indicative of memory or attentional difficulties, for example. Finally, including sleep-related treatments (described below) in such a package approach may be warranted based on the significant sleep-related problems associated with PTSD.

*Treatment of sleep-related symptoms*

The biological research on sleep and PTSD suggests that sleep-related symptoms are significant in this population and likely result in additional difficulties (e.g., problems with concentration, motivation, and interpersonal relationships) for patients. Whereas some individuals with PTSD may also meet criteria for a diagnosis of primary insomnia, sleep-related difficulties more frequently occur secondary to the hyperarousal and re-experiencing symptoms associated with PTSD. Primary insomnia in adults is conceptualized as a function of inappropriate situational cues for sleeping, excessive muscle tension, and excessive sleep-related worry based on erroneous or distorted beliefs [65]. Even when sleep-related symptoms occur secondary to PTSD, we recommend that empirically supported interventions used in the treatment of primary insomnia be incorporated into the treatment of individuals with PTSD who demonstrate problems with initiating or maintaining sleep. Such treatment approaches would include improving sleep hygiene, increasing appropriate bedtime stimulus control, sleep restriction to decrease the hours spent in bed to include only those spent sleeping, sleep compression to gradually delay time entering bed and advance morning arising time, and various relaxation methods [66]. Many relaxation methods have been implemented in the treatment of insomnia and include progressive muscle relaxation, autogenic training, biofeedback, and meditation [67].

Although direct treatment of insomnia symptoms may be helpful for individuals with PTSD, sleep is often disrupted by nightmares in this population. For this reason, directly targeting the symptom of nightmares may be especially important in improving the quality of sleep for these individuals. In addition to some of the relaxation techniques described above, another intervention for reducing nightmares with some empirical support is imagery rehearsal. In this exposure-based intervention, the individual rehearses a changed version of the recurrent nightmare using cognitive imagery [68]. This is typically a short intervention (one to four sessions) that is effective for

chronic nightmare sufferers [69], veterans with combat-related PTSD [68], and sexual assault survivors with PTSD [70]. Given the simplicity and brevity of this intervention, treatment of nightmares associated with PTSD should be considered in comprehensive treatment approaches whenever possible.

### *Innovative treatment approaches for PTSD*

#### *Acceptance and commitment therapy*

Acceptance and Commitment Therapy (ACT) is a behavior therapy approach that has been successfully applied to the treatment of a variety of disorders involving experiential avoidance [71]. Experiential avoidance is a process that occurs when a person is unwilling to experience unwanted thoughts, feelings, or memories; the individual thus takes action to change the form, frequency, or contexts evoking these private experiences. There is a growing body of evidence supporting the role of experiential avoidance in the development and maintenance of PTSD, and, in fact, avoidant symptoms comprise one of the three main clusters that define PTSD. Furthermore, research suggests that avoidance of private events often results in a subsequent rebound of the avoided thoughts and feelings [71], and thus the re-experiencing symptoms of PTSD may actually be exacerbated by avoidance. At its core, ACT works to help clients decrease ineffective patterns of behavior, reduce the use of experientially avoidant strategies, and initiate commitments to action that are consistent with life directions valued by the individual. Randomized clinical trials support the use of ACT in the treatment of depression, psychotic symptoms, workplace stress, and substance abuse, with preliminary evidence for chronic pain, anorexia, GAD, and other anxiety disorders [72].

ACT has clear implications for the treatment of PTSD because of the core features of avoidance and re-experiencing in the disorder. An ACT approach to the treatment of PTSD would begin with an analysis of the strategies that the individual uses to manage his/her problems and symptoms while identifying the role that avoidance has played in trying to numb or reduce psychological pain and difficult memories. The ineffectiveness of existing strategies is illuminated, and through metaphor and experiential exercises, clients are asked to work on their willingness to experience whatever thoughts, feelings, or memories are present, in the service of moving forward in their lives. In addition to the metaphor and experiential work that are characteristic of ACT, clients with PTSD may also be asked to discuss their traumatic events in detail, being willing to feel and remember whatever is present for them. To an untrained observer, this work often looks like the work of exposure therapy; however, there is one significant difference between the two approaches. Unlike exposure therapy, ACT does not assume that processing painful emotions and memories will necessarily lead to an eventual reduction in emotional pain caused by extinction. Although painful emotions associated with a trauma often do decrease over

the course of therapy, the ACT approach views willingness to experience cognitions and emotions associated with traumatic experiences as pivotal. Most importantly, the emphasis is on encouraging the patient to move forward with his or her life, which is viewed as a more workable goal than a total reduction in symptoms. The use of ACT for PTSD awaits further empirical validation but is a promising direction for future treatment development.

The biological findings reviewed earlier also support this approach. In particular, the ERP data suggest that PTSD patients' tendency to shut out new stimuli may represent learned avoidance. The focus of ACT on the reduction of avoidance may help patients to respond to stimuli on the basis of their actual characteristics rather than with a response of avoidance or hyperarousal. Over time, experience with the actual consequences of internal and external stimuli may result in extinction of emotional responding and a decreased need for hypervigilance. In addition, those who do not respond or are not willing to engage in exposure therapy may be those with the highest levels of avoidance. ACT may help to engage such individuals into a treatment that involves emotional processing when direct exposure treatment is not feasible. Finally, given the implications cited above for the use of visual cues caused by reductions in hippocampal volume, ACT's use of metaphors and visual imagery may be especially useful for individuals with PTSD. Although clinical trials demonstrating its value in treating PTSD are needed, the availability of an alternative treatment to exposure therapy that is also based on an avoidance model of psychopathology is particularly welcome at this time.

*Interpersonal psychotherapy*

Interpersonal psychotherapy (IPT) is a treatment approach originally developed by Klerman et al. for the treatment of depression [73] and is borne out of psychodynamic tradition. The IPT approach emphasizes an individual's difficulties in interpersonal relations, which may include interpersonal deficits, disputes, role transitions, and bereavement/loss [74]. IPT is an equally effective treatment approach for major depression when compared with CBT and pharmacotherapy [75]. Recently, IPT was adapted as a short-term (16-session) group treatment for low-income women with PTSD related to an interpersonal trauma (e.g., rape, child sexual or physical abuse, domestic abuse) (Janice Krupnick, PhD, personal communication, 2001). IPT presumes that interpersonal trauma may cause relationship problems that result in either an individual remaining out of relationships or, alternatively, re-enacting prior abuses in current relationships. The therapy focuses on increasing the patient's understanding of how current relationship patterns have been affected by past trauma and PTSD. The treatment approach uses out-of-therapy relationships and in-therapy reactions as opportunities to explore these behavior patterns. Its treatment targets include reduction of revictimization and the social isolation associated with the disorder.

Because PTSD is clearly associated with difficulties in interpersonal relationships, this treatment approach has much promise and is currently being evaluated by Krupnick and colleagues. As noted by McFarlane and Yehuda [76], some trauma survivors seek treatment to "repair the disruption of relationships due to interpersonal estrangement" (p. 949). These authors argue that focusing on the traumatic event may not be effective for all patients diagnosed with PTSD. Also, research on empirically validated approaches (e.g., exposure therapy) indicates that not all patients respond to treatments with the most empirical support [55]. No studies to date have demonstrated IPT's effectiveness for the treatment of PTSD, although research by Brom et al. provides preliminary support for a short-term, individual, dynamically oriented treatment approach for PTSD [77]. If IPT's effectiveness is supported by current research, future work into its applicability for other traumatized populations (e.g., combat veterans) will be warranted. Additionally, on the basis of the biological findings reviewed earlier suggesting possible subtypes of the disorder, IPT may be particularly well suited for individuals who demonstrate less of a fear-based response, more shame and guilt, or fewer re-experiencing symptoms. This hypothesis can be tested and will increase our understanding of the variables associated with treatment success or failure. Finally, the biological research suggesting that victims of a prior rape or assault respond differently to a subsequent assault [20] suggests that IPT's emphasis on relationship patterns that may place individuals at risk for revictimization may be particularly important in the prevention of subsequent exposure and the eventual development of PTSD.

*Early intervention and prevention of revictimization*

Whereas most treatments developed for PTSD have focused on amelioration of chronic symptoms, an important direction for future study is intervention in the early aftermath of exposure. Although the direction of the relationship between PTSD and associated biological findings is largely unknown, it is reasonable to conclude that one of the best ways to prevent some of the disruption associated with long-term PTSD is to prevent the initial development of PTSD after a potentially traumatizing event. Additionally, research on cortisol and heart rate levels immediately following a traumatic event suggests that these may be biological markers for the development of PTSD that could be useful in identifying individuals that might benefit from a targeted preventative treatment. Although this area of research remains in its infancy and previous results on early intervention have been equivocal [78], there does exist preliminary evidence for the utility of psychological interventions in the prevention of PTSD. Foa et al. [12] found that recent rape victims who were given a treatment package consisting of exposure therapy, education, breathing retraining, and cognitive restructuring were significantly less likely to develop PTSD 2 months post-intervention than the untreated comparison group. Similarly, Bryant et al. [13] found that a treatment package consisting of exposure therapy, cognitive

restructuring, and anxiety management for acute stress disorder (i.e., the precursor of PTSD) resulted in a significant reduction in the number of individuals who met criteria for PTSD at 6-month follow-up, compared with those receiving supportive counseling. Furthermore, given the biological data suggesting that a history of traumatic events may put one at risk for developing PTSD after additional exposures, the prevention of symptomatology in persons who have experienced a traumatic event is a public health priority. More work also needs to address the development and evaluation of brief psychological interventions [79,80] following trauma exposure in the prevention of revictimization.

## Conclusion

Although we do not have a comprehensive understanding of the psychological and biological processes underlying PTSD, significant progress in these areas is forthcoming. As new technologies emerge, better research methods are used, and more sophisticated analyses are conducted, additional progress in our understanding of this complex disorder will certainly be made. At this juncture, it is best that we look for clues to these underlying parameters when developing treatment programs. Presently, we do not have a complete functional analysis of the problems associated with PTSD. Information on the biological, psychological, and behavioral defects associated with PTSD will contribute to the continuing development and refinement of psychological treatments. Inversely, an analysis of the strengths and assets of individuals with PTSD will further direct us to new treatment methods. Fundamental progress will be made in the psychological treatment of PTSD when we have an analysis of those functions that are compromised and those spared when PTSD evolves following trauma.

In the development of psychological treatments, it is valuable to appreciate the possibility of subtypes of this condition. These subtypes may require different treatments or different combinations of treatments to optimize psychosocial outcomes. Moreover, there is a need for new, empirically validated treatments that match the backgrounds of both the patients *and* the therapists. Having more treatment options from which therapists can choose would further advance the field of PTSD.

Future work on the biological and psychological parameters associated with PTSD should include prospectively designed studies with a particular emphasis on the integration of biological and psychological markers. Crossing levels of analysis from the genetic to the cellular to the systemic to the behavioral level is likely to result in rapid advances in our knowledge base. From this enhanced knowledge base will come future improvements in psychological treatments.

In addition, for future advances in the psychological treatment of PTSD, psychological researchers would benefit from a more complete understanding of the biological correlates of the disorder. Similarly, those interested in

the biology of PTSD would enhance the field by attending to the measurement and analysis of relevant psychological variables. This would minimize the over-reliance on a mechanistic view of this dynamic psychological condition. Finally, as a field, we would benefit from increased collaboration among all those committed to a scientific, multilevel analysis of PTSD with the goal of developing empirically supported, comprehensive treatments for PTSD—treatments that are based upon a scientific understanding of this disorder. This approach to treating PTSD will likely alleviate the suffering associated with exposure to traumatic events and the development of PTSD.

## References

[1] Foa EB, Keane TM, Friedman MJ. Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies. New York: Guilford Press; 2000.
[2] Kardiner A. The traumatic neuroses of war 1941New York: Hoeber.
[3] Keane TM, Kolb LC, Kaloupek DG, Orr SP, Blanchard EB, Thomas RG, et al. Utility of psychophysiological measurement in the diagnosis of post-traumatic stress disorder: results from a Department of Veterans Affairs Cooperative Study. J Consult Clin Psychol 1998;66: 914–23.
[4] Orr SP, Lasko NB, Metzger LJ, Berry NJ, Ahern CJ, Pitman RK. Psychophysiologic assessment of women with posttraumatic stress disorder resulting from childhood sexual abuse. J Consult Clin Psychol 1998;66:906–13.
[5] Blanchard EB, Hickling EJ, Buckley TC, Taylor AE, Vollmer A, Loos WR. Psychophysiology of posttraumatic stress disorder related to motor vehicle accidents: replication and extension. J Consult Clin Psychol 1996;64:742–51.
[6] Prins A, Kaloupek DG, Keane TM. Psychophysiological evidence for autonomic arousal and startle in traumatized adult populations. In: Friedman MJ, Charney DS, Deutch AY, editors. Neurobiological and clinical consequences of stress: from normal adaptation to post-traumatic stress disorder. Philadelphia: Lippincott-Raven; 1995. p. 291–314.
[7] Griffin MG, Resick PA, Mechanic MB. Objective assessment of peritraumatic dissociation: psychophysiological indicators. Am J Psychiatry 1997;154:1081–8.
[8] Blanchard EB. Elevated basal levels of cardiovascular responses in Vietnam veterans with PTSD: a health problem in the making? J Anxiety Dis 1990;4:233–7.
[9] Pitman RK, Orr S, Forgue D, Altman JB, deJong JB, Herz R. Psychophysiologic responses to combat imagery of Vietnam veterans with posttraumatic stress disorder versus other anxiety disorders. J Abnorm Psychol 1990;99:49–54.
[10] Buckley TC, Kaloupek DG. A meta-analytic examination of basal cardiovascular activity in PTSD. Psychosom Med, in press.
[11] Shalev AY, Sahar T, Freedman S, Peri T, Glick N, Brandes D, et al. A prospective study of heart rate response following trauma and the subsequent development of posttraumatic stress disorder. Arch Gen Psychiatry 1998;55:553–9.
[12] Foa EB, Hearst-Ikeda D, Perry KJ. Evaluation of a brief cognitive-behavioral program for the prevention of chronic PTSD in recent assault victims. J Consult Clin Psychol 1995;63:948–55.
[13] Bryant RA, Harvey AG, Dang ST, Sackville T, Basten C. Treatment of acute stress disorder: a comparison of cognitive-behavioral therapy and supportive counseling. J Consult Clin Psychol 1998;66:862–6.
[14] Chrousos GP, Gold PW. The concepts of stress and stress system disorders: overview of physical and behavioral homeostasis. JAMA 1992;267:1244–52.
[15] Yehuda R, Teicher M, Trestman RL, Levengood RA, Siever RJ. Cortisol regulation in post-traumatic stress disorder: a chronobiological analysis. Ann NY Acad Sci 1994;746:378–80.

[16] Yehuda R, Lowy MT, Southwick SM, Shaffer D, Giller EL. Increased number of glucocorticoid receptor number in post-traumatic stress disorder. Am J Psychiatry 1991; 149:499–504.
[17] Pitman RK, Orr SP. Twenty-four hour urinary cortisol and catecholamine excretion in combat-related posttraumatic stress disorder. Biol Psychiatry 1990;27:245–7.
[18] Yehuda R. Sensitization of the hypothalamic-pituitary-adrenal axis in post-traumatic stress disorder. Ann NY Acad Sc 1997;821:57–75.
[19] Yehuda R, McFarlane AC, Shalev AY. Predicting the development of post-traumatic stress disorder from the acute response to a traumatic event. Biol Psychiatry 1998;44:1305–13.
[20] Resnick H, Yehuda R, Pitman R, Foy D. Effect of previous trauma on acute plasma cortisol level following rape. Am J Psychiatry 1995;152:1675–7.
[21] Meany MJ, Aitken DH, Viau V, Sharma S, Sarrieau A. Neonatal handling alters adrenocorticol negative feedback sensitivity and hippocampal type II glucocorticoid receptor binding in the rat. Neuroendocrinology 1989;50:597–604.
[22] Southwick SM, Paige S, Morgan III A, Bremner JD, Krystal JH, Charney DS. Neurotransmitter alterations in PTSD: catecholamines and serotonin. Semin Clin Neuropsychiatry 1999;4:242–8.
[23] Kosten TR, Mason JW, Giller EL, Ostroff RB, Harkness L. Sustained urinary norepinephrine and epinephrine elevation in post-traumatic stress disorder. Psychoneuroendocrinology 1987;12:13–20.
[24] Perry BD, Giller EL, Southwick SM. Altered platelet alpha2 adrenergic binding sites in post-traumatic stress disorder. Am J Psychiatry 1987;144:1511–2.
[25] Southwick SM, Krystal JH, Morgan AC, Johnson D, Nagy L, Nicolaou A, et al. Abnormal noradrenergic function in posttraumatic stress disorder. Arch Gen Psychiatry 1993;50:266–74.
[26] Yehuda R, Perry BD, Southwick SM, Giller EL. Platelet alpha2 adrenergic binding in PTSD, generalized anxiety disorder and major depressive disorder. In: New research abstracts, 143. New York: American Psychiatric Association; 1990.
[27] Watanabe Y, Gould E, McEwen BS. Stress induces atrophy of apical dendrites of hippocampal CA3 pyramidal neurons. Brain Res 1992;588:341–4.
[28] Bremner JD, Randall P, Scott TM, Bronen RA, Seibyl JP, Southwick SM, et al. MRI-based measures of hippocampal volume in patients with PTSD. Am J Psychiatry 1995;152: 973–81.
[29] Gurvits TV, Shenton ME, Hokama H, Ohta H. Magnetic resonance imaging study of hippocampal volume in chronic combat-related posttraumatic stress disorder. Biol Psych 1996;40:1091–9.
[30] Bremner JD, Randall P, Vermetten E, Staib L, Bronen RA, Mazure C, et al. Magnetic resonance imaging-based measurement of hippocampal volume in posttraumatic stress disorder related to childhood physical and sexual abuse: a preliminary report. Biol Psychiatry 1997;41:23–32.
[31] Stein MB, Koverola C, Hanna C, Torchia MG, McClarty B. Hippocampal volume in women victimized by childhood sexual abuse. Psychol Med 1997;27:951–9.
[32] De Bellis MD, Keshavan MS, Clark DB, Casey BJ, Giedd JN, Boring AM, et al. Developmental traumatology. Part II: Brain development. Biol Psychiatry 1999;45:1271–84.
[33] Paige SR, Reid GM, Allen MG, Newton J. Psychophysiological correlates of posttraumatic stress disorder in Vietnam veterans. Biol Psychiatry 1990;27:419–30.
[34] Gillette GM, Skinner RD, Rasco LM, Fielstein E, Davis D, Pawela KJ, et al. Combat veterans with posttraumatic stress disorder exhibit decreased habitation of the P1 mid-latency auditory evoked potential. Life Sci 1997;61:1421–34.
[35] Neylan TC, Fletcher DJ, Lenoci M, McCallin K, Weiss DS, Schoenfeld FB, et al. Reduced auditory P50 suppression in combat veterans. Biol Psychiatry 1999;46:1656–64.
[36] Attias J, Bleich A, Firman V, Zinger Y. Event-related potentials in post-traumatic stress disorder of combat origin. Biol Psychiatry 1996;40:373–81.

[37] Ross RJ, Ball WA, Dinges DF, Kribbs NB, Morrison AR, Silver SM, et al. Rapid eye movement sleep disturbance in posttraumatic stress disorder. Biol Psychiatry 1994;35: 195–202.
[38] Mellman TA, Nolan B, Hebding J, Kulick-Bell R, Dominguez R, et al. A polysomnographic comparison of veterans with combat-related PTSD, depressed men, and non-ill controls. Sleep 1997;20:46–51.
[39] Lavie P, Katz N, Pillar G, Zinger Y. Elevated awakening thresholds during sleep: characteristics of chronic war-related posttraumatic stress disorder patients. Biol Psychiatry 1998;44:1060–5.
[40] Mellman TA. Psychobiology of sleep disturbances in posttraumatic stress disorder. Ann NY Acad Sci 1997;821:142–9.
[41] Dagan Y, Lavie P, Bleich A. Elevated awakening thresholds in sleep stage, 3–4 in war-related post-traumatic stress disorder. Biol Psychiatry 1991;30:618–22.
[42] David D, Mellman A. Dreams following Hurricane Andrew. Presented at the 148th Annual Meeting of the American Psychiatric Association. Miami, FL, 1995.
[43] Black JL, Keane TM. Implosive therapy in the treatment of combat related fears in a World War II veteran. J Behav Ther Exp Psychiatry 1982;13:163–5.
[44] Fairbank JA, Keane TM. Flooding for combat-related stress disorders: Assessment of anxiety reduction across traumatic memories. Beh Ther 1982;13:499–510.
[45] Keane TM, Kaloupek DG. Imaginal flooding in the treatment of posttraumatic stress disorder. J Consult Clin Psychol 1982;50:138–40.
[46] Keane TM, Fairbank JA, Caddell JM, Zimering RT, Bender ME. Implosive (flooding) therapy reduces symptoms of PTSD in Vietnam combat veterans. Behav Ther 1989;20:245–60.
[47] Boudewyns PA, Hyer L. Physiological response to combat memories and preliminary treatment outcome in Vietnam veteran PTSD patients treated with direct therapeutic exposure. Behav Ther 1990;21:63–87.
[48] Cooper NA, Clum GA. Imaginal flooding as a supplementary treatment for PTSD in combat veterans: A controlled study. Behav Ther 1989;20:381–91.
[49] Foa EB, Rothbaum BO, Riggs DS, Murdock TB. Treatment of posttraumatic stress disorder in rape victims: A comparison between cognitive-behavioral procedures and counseling. J Consult Clin Psychol 1991;59:715–23.
[50] Marks I, Lovell K, Noshirvani H, Livanou M, Thrasher S. Treatment of posttraumatic stress disorder by exposure and/or cognitive restructuring: A controlled study. Arch Gen Psychiatry 1998;55:317–25.
[51] Richards DA, Lovell K, Marks IM. Post-traumatic stress disorder: Evaluation of a behavioral treatment program. J Traum Stress 1994;7:669–80.
[52] Tarrier N, Pilgrim H, Sommerfield C, Faragher B, Reynolds M, Graham E, et al. A randomized trial of cognitive therapy and imaginal exposure in the treatment of chronic posttraumatic stress disorder. J Consult Clin Psychol 1999;67:13–8.
[53] Foa EB, Dancu DV, Hembree EA, Jaycox LH, Meadows EA, Street GP. A comparison of exposure therapy, stress inoculation training, and their combination for reducing posttraumatic stress disorder in female assault victims. J Consult Clin Psychol 1999;67:194–200.
[54] Keane TM, Barlow DH. Posttraumatic stress disorder. In: Barlow DH, editor. Anxiety and its disorders: the nature and treatment of anxiety and panic. New York: Guilford; in press.
[55] Jaycox LH, Foa EB. Obstacles in implementing exposure therapy for PTSD: case discussions and practical solutions. Clin Psychol Psychother 1996;3:176–84.
[56] Chemtob CM, Novaco RW, Hamada RS, Gross DM. Cognitive behavioral treatment of severe anger in posttraumatic stress disorder. J Consult Clin Psychol 1997;65:184–9.
[57] Peniston EG. EMG biofeedback-assisted desensitization treatment for Vietnam combat veterans with post-traumatic stress disorder. Clin Biofeedback Health 1986;9:35–41.
[58] Keane TM, Fisher LM, Krinsley KE, Niles BL. Posttraumatic stress disorder. In: Hersen M, Ammerman RT, editors. Handbook of prescriptive treatments for adults. New York: Plenum; 1994. p. 237–60.

[59] Fecteau G, Nicki R. Cognitive behavioral treatment of post-traumatic stress disorder after motor vehicle accident. Behav Cog Psychother 1999;27:201–14.
[60] Frueh BC, Turner SM, Beidel DC, Mirabella RF, Jones WJ. Trauma Management Therapy: a preliminary evaluation of a multicomponent behavioral treatment for chronic combat-related PTSD. Behav Res Ther 1996;34:533–43.
[61] Resick PA, Schnicke MK. Cognitive processing therapy for sexual assault victims. J Consult Clin Psychol 1992;60:748–56.
[62] Resick PA, Nishith P, Astin M. A comparison of cognitive processing therapy and prolonged exposure: a randomized controlled clinical trial. Paper presented at the World Conference of the International Society for Traumatic Stress Studies. Melbourne, Australia, 2000.
[63] Shapiro F. Eye movement desensitization: a new treatment for post-traumatic stress disorder. J Behav Ther Exp Psychiatry 1989;20:211–7.
[64] Shapiro F. Eye movement desensitization and reprocessing: basic principles, protocols, and procedures 1995;20:New York: Guilford.
[65] Spiegler MD, Guevremont DC. Contemporary behavior therapy. 3rd edition. New York: Brooks/Cole; 1998.
[66] Van Brunt DL, Lichstein KL. Primary insomnia. In: Hersen M, Biaggio M, editors. Effective brief therapies: a clinician's guide. New York: Academic Press; 2000. p. 283–302.
[67] Lichstein KL, Fischer SM. Insomnia. In: Hersen M, Bellack AS, editors. Handbook of clinical behavior therapy with adults. New York: Plenum; 1985. p. 319–52.
[68] Thompson KE, Hamilton M, West JA. Group treatment for nightmares in veterans with combat-related PTSD. National Center for PTSD Clinical Quarterly 1995;5:13–7.
[69] Neidhardt EJ, Krakow B, Kellner R, Pathak D. The beneficial effects of one treatment session and recording of nightmares on chronic nightmare sufferers. Sleep 1992;15: 470–3.
[70] Krakow B, Hollifield M, Johnston L. Imagery rehearsal therapy for chronic nightmares in sexual assault survivors with posttraumatic stress disorder: a randomized controlled trial. JAMA 2001;286:537–45.
[71] Hayes SC, Strosahl KD, Wilson KG. Acceptance and Commitment Therapy: an experiential approach to behavior change. New York: Guilford; 1999.
[72] Hayes SC, Pankey J, Batten SV, Gifford EV, Quinones R. Acceptance and Commitment Therapy in the treatment of experiential avoidance disorders. In: Patterson T, editor. Comprehensive handbook of psychotherapy. Volume 2: Cognitive/behavioral/functional approaches. New York: Wiley; in press.
[73] Klerman GL, Weissman MW, Rounsaville BJ, et al. Interpersonal psychotherapy of depression 1984;Northvale, NJ: Jason Aronson.
[74] Krupnick JL. Interpersonal psychotherapy for PTSD following interpersonal trauma. Directions Psychiatry 2000;20:237–52.
[75] Elkin I, Shea MT, Watkins JT, Imber SD, Sotsky SM, Collins JF, et al. NIMH Treatment of Depression Collaborative Research Program: general effectiveness of treatments. Arch Gen Psychiatry 1989;46:971–83.
[76] McFarlane AC, Yehuda R. Clinical treatment of posttraumatic stress disorder: conceptual challenges raised by recent research. Aust N Z J Psychiatry 2000;34:940–53.
[77] Brom D, Kleber RJ, Defares PB. Brief psychotherapy for posttraumatic stress disorders. J Consult Clin Psychol 1989;57:607–12.
[78] Kenardy JA, Webster RA, Lewin TJ, Carr VJ, Hazell PL, Carter GL. Stress debriefing and patterns of recovery following a natural disaster. J Traum Stress 1996;9:37–49.
[79] Hanson KA, Gidycz CA. Evaluation of a sexual assault prevention program. J Consult Clin Psychol 1993;61:1046–52.
[80] Marx BP, Calhoun KS, Wilson AE, Meyerson LA. Sexual revictimization prevention: an outcome evaluation. J Consult Clin Psychol 2001;69:25–32.